The Relationship Between Epicardial Adipose Tissue Volume and Coronary Plaque Structure in Diabetics

Diyabetiklerde Epikardiyal Yağ Volümü ile Koroner Plak Yapısı Arasındaki İlişki

Epicardial Adipose Tissue and Coronary Plaque

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Özet

Amaç: Epikardiyal yağ dokusu (EYD) koroner arter hastalığı (KAH) gelişimi ve ilerlemesinde rol oynamaktadır. Biz bu çalışmamızda, diyabetik hastalarda EYD volümü ile koroner ateroskleroz, koroner plak yükü ve plak yapısı arasındaki ilişkiyi incelemeyi amaçladık. Gereç ve Yöntem: KAH şüphesi araştırılması için 128 kesitli bilgisayarlı tomografik koroner anjiografi ile değerlendirilen 196 diyabetes mellitus (DM) hastası çalışmaya dahil edildi. Toplam plak yükü, hasta damar segmenti sayısı, plak karakteristikleri ve EYD volümü BT anjiografi ile değerlendirildi. Hastalar iki gruba ayrıldı [KAH olan grup (Grup I) ve KAH olmayan grup (Grup II)]. Her segmentte plak karakteristikleri ayrı ayrı analiz edildi. Bulgular: EYD volümü, KAH olan DM hastalarında KAH olmayan DM hastalarına göre anlamlı derecede yüksek bulundu (138.7±49.1 ml vs 98.6±34.7 ml, p<0.001). Korelasyon analizinde EYD volümü, vücut kitle indeksi (VKİ) (r=0.369, p<0.001), toplam plak yükü (r=0.424, p<0.001), miks plak (r=0.454, p<0.001), kalsifik olmayan plak (r=0.369, p<0.001), kalsifik plak (r=0.191, p=0.007) ve hasta damar segmenti sayısı (r=0.449, p<0.001) ile anlamlı pozitif korelasyon göstermekte idi. Ayrıca, multivaryant lojistik regresyon analizinde, EYD volümünün DM hastalarında KAH varlığı için önemli ve bağımsız bir prediktör olduğu saptandı (OR=1.023, 95% CI: 1.014-1.032; p<0.001). Tartışma: DM hastalarında KAH varlığı için EYD volümünün bağımsız bir prediktör olduğunu saptadık. Dahası, EYD volümü toplam plak yükü ve miks ve kalsifik olmayan plak sayısı ile orta derece korelasyon göstermekte iken, kalsifik plak sayısı ile zayıf korelasyon göstermekte idi.

Anahtar Kelimeler

Epikardiyal Yağ Dokusu; Koroner Arter Hastalığı; Koroner Plak

Abstract

Aim: Epicardial adipose tissue (EAT) contributes to the development and progression of coronary artery disease (CAD). We aimed to evaluate the relationship between EAT volume, coronary atherosclerosis, coronary plaque burden, and plaque structure in diabetic patients. Material and Method: 196 DM patients who were evaluated with 128-slice dual- source coronary computed tomography angiography (CCTA) for suspected CAD were included in the study. The CCTA examination was used to assess the total plaque burden, number of diseased segments, plaque characteristics, and EAT volume. The study population was divided into two groups [a CAD group (Group I) and non-CAD group (Group II)]. The plaque characteristics were analyzed on a per-segment basis. Results: EAT volume was found to be significantly higher among diabetic patients with CAD compared to those without CAD (138.7±49.1 ml vs 98.6±34.7 ml, p<0.001). In the correlation analysis, EAT volume showed significant positive correlation with BMI (r=0.369, p<0.001), total plaque burden (r=0.424, p<0.001), mixed plaques (r=0.454, p<0.001), non-calcified plaques (r=0.369, p<0.001), calcified plaques (r=0.191, p=0.007), and number of diseased segments (r=0.449, p<0.001). Also, multivariate logistic-regression analysis revealed that EAT volume to be a significant and independent predictor of the presence of CAD in patients with DM (OR=1.023, 95% CI: 1.014-1.032; p<0.001). Discussion: We have determined that EAT volume is an independent predictor among diabetic patients for the presence of CAD. Moreover, EAT volume showed moderate correlation with total plaque burden and the number of mixed and non-calcified plaques but weak correlation with calcified plaques.

Keywords

Epicardial Adipose Tissue; Coronary Artery Disease; Coronary Plaque

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Introduction

Diabetes mellitus (DM) is considered to be an important risk factor for cardiovascular diseases. Chronic inflammation and oxidative stress, which play key roles in the development of atherosclerotic disease, are increased in diabetic patients [1]. As morbidity and mortality increase due to coronary artery disease (CAD) in diabetic patients, many studies have focused on markers that can predict CAD and can provide prognostic information about diabetics.

Epicardial adipose tissue (EAT) is the visceral fat tissue that surrounds the heart and coronary arteries. By secreting various pro- and anti-inflammatory cytokines and chemokines by endocrine and paracrine activity, EAT contributes to the coronary atherosclerosis process [2]. Studies have shown that EAT is increased in CAD, and associated with the presence and extent of CAD [3-4]. There are also reports that EAT may be a predictor for future cardiovascular events [5]. A study that investigated the association of EAT with CAD in diabetic patients revealed its association with the presence and extent of CAD [6].

The first method used to measure EAT thickness was echocardiography. However, methods that allow for extensive volumetric measurements such as magnetic resonance imaging (MRI) and multidetector computed tomography (MDCT) are preferred today. Accordingly, EAT volume measured by MDCT shows greater reproducibility than echocardiographically-measured EAT thickness [7]. Additionally, knowledge of the degree of coronary artery stenosis and the structure of coronary plaque with MDCT provides extra benefits [8].

Although the relationship between EAT volume and CAD is well documented, there are no data concerning the relationship between EAT volume and coronary plaque burden and structure in diabetic patients.

Therefore, we aimed to evaluate the association between EAT volume, coronary atherosclerosis, and coronary plaque burden and structure in diabetics.

Material and Method

Study population

This retrospective analysis was performed in a subset of 842 patients who were admitted into our Cardiology department for cardiovascular evaluation between April 2009 and February 2016 and in whom coronary computed tomography angiography (CCTA) was performed for suspicion of CAD after clinical assessment. 196 diabetic patients among this group were included in the study. The indications for CCTA in the study population were atypical chest pain with an intermediate risk for CAD, inconclusive or interpretable stress test result, suspected coronary anomalies, and exclusion of CAD among patients undergoing noncoronary cardiac surgery. After an evaluation of the CCTA images, the study population was divided into two groups [CAD group (Group I) and non-CAD group (Group II)] on the basis of the presence of coronary plaques. Missing information regarding demographic and clinical features of the patients and their medications was supplied through evaluation of patient records.

Patients with previously documented CAD, acute coronary syndrome (ACS), percutaneous coronary intervention, coronary bypass surgery, heart failure, renal disease, malignancy, active or chronic inflammatory diseases, and hepatic dysfunction were excluded from the study.

Diabetes mellitus was defined as a fasting plasma glucose level >126 mg/dl or hemoglobin A1c (HbA1c) >6.5% or current treatment with insulin or oral hypoglycemic agents. Hypertension (HT) was defined as systolic blood pressure >140 mmHg or diastolic blood pressure >90 mmHg or treatment with antihypertensive drugs.

Laboratory measurements

Relevant laboratory test results that had been measured at the time of cardiovascular evaluation (before CCTA procedures) were obtained from medical reports. Hemoglobin, platelet values, and white blood cell count were obtained from the automated CBC using a Sysmex XE-2100 Automated Hematology Blood Analyzer System (Sysmex, Kobe, Japan). Biochemical parameters were measured with a Cobas 8000 Modular Analyzer (Roche Diagnostics, Indianapolis, USA) using commercially available assay kits.

Coronary computed tomography angiography: image acquisition

All scans were performed using a 128-slice dual source computed tomography system. (Somatom Definition AS; Siemens Healthcare, Forchheim, Germany). Patients with an initial heart rate of ≥65 beats/min were given β blocker therapy according to the local protocols. Every participant received 0.4 mg of sublingual nitroglycerin 1 minute prior to contrast administration in order to dilate coronary arteries. The coronary angiographic scan was obtained with injection of 70-80 ml nonionic contrast medium (350 mg I/ml iomeprol; Bracco Omnipaque, Milano, Italy) at a flow rate of 5 mL/s followed by 50 mL of saline solution, and contrast administration was controlled with bolus tracking. The acquisition parameters were 2x64x0.6 mm detector collimation, resulting in 2x128x0.6 mm sections by means of the z-axis flying focal spot technique, 280 ms gantry rotation time, 75 ms temporal resolution, 100 to 120 kV tube voltage depending on body mass index (no greater than 30 kg/m2 BMI, 100 kV; greater than 30 kg/m2 BMI, 120 Kv), 330 mAs per rotation tube current and 0.2-0.47 pitch adapted to the heart rate. An ECG-gated helical mode scan was performed with the full radiation dose window set at 68-78% of the R-R interval in patients with heart rates ≤70 bpm or 200-400 ms after the R peak in patients with heart rate of >70 bpm. The minimum tube current with 4% of the full radiation dose (MinDose; Siemens Healthcare, Forchheim, Germany) was applied to the remainder of the R-R interval to minimise radiation dose. Images were reconstructed with a slice thickness of 0.6 mm, a reconstruction increment of 0.4 mm, and using a soft-tissue convolution kernel (B26f).

Coronary computed tomography angiography: image analysis

All scans were analyzed independently by two experienced radiologists who were blinded to the clinical information, using a 3D workstation (Syngo; Siemens Healthcare, Erlangen, Germany). After making independent evaluations, a consensus interpretation was arrived at to obtain a final CCTA diagnosis. Each identified lesion was examined using maximum intensity projection and multiplanar reconstruction techniques on short axis and along multiple longitudinal axes. The radiologists analyzed

the plaque characteristics on a per-segment basis according to the modified American Heart Association classification [9]. Plaques were defined as structures >1 mm2 within and/or adjacent to the vessel lumen, which could be clearly distinguished from the lumen and surrounding pericardial tissue. Coronary plaques were classified as non-calcified, calcified, and mixed according to their structure. Plaques without any calcification were defined as non-calcified plaques, plaques with more than 50% of the plaque area occupied by calcified tissue (density ≥130 HU in native scans) were defined as calcified, and plaques with less than 50% calcified tissue were defined as mixed type [10]. All plaque components were assessed on a per-segment basis. The number of all plaques (total plaque burden), as well as plaques with different features, were calculated per patient. Inter-observer agreement for the detection of any plaque/patient and plaque/segment was excellent (Cohen's κ=0.91 and 0.83, respectively).

Epicardial adipose tissue volume measurement

EAT was defined as the adipose tissue within the visceral epicardium. The layer of epicardium was manually traced from the mid-left atrium to the left ventricular apex with 10 mm thick spacing between each image; all extrapericardial tissue was excluded. The computer software automatically constructed a three-dimensional image of the epicardium. EAT volume was quantified by calculating the total volume of the tissue whose CT density ranged between -250 HU and -30 HU within the epicardium [3]. This range can effectively exclude myocardium, coronary arteries, coronary calcium, and blood pool. Measurements were done separately on a workstation (Leonardo, Siemens Healthcare, Germany) with dedicated software (Syngo, Siemens Healthcare, Germany). The reproducibility of the EAT volume measurements was evaluated in a random sample of 24 participants and the intra- and inter-observer reproducibilities for EAT volume were excellent (correlation coefficients 0.98 and 0.97, respectively).

Statistical analysis

All data were loaded to the SPSS 15 program. The normal distribution of the data was tested using the Kolmogorov–Smirnov test. Logarithmic transformation was performed on all non-normally distributed variables. The Student's t test was used to compare two groups of values. Comparison of categorical values was carried out by the chi-square test. Any correlation between data was tested with the Spearman correlation analysis. Multivariate logistic-regression analysis was also performed, and the model included the potential confounders (age, male sex, body mass index, serum creatinine, low-density lipoprotein, high-density lipoprotein, HbA1c, hypertension, smoking, epicardial adipose tissue volume). While the continuous data were expressed with 'mean ± SD (standard deviation)', the categorical data were expressed with percentage values and a p value of <0.05 was accepted as statistically significant.

Results

Baseline clinical characteristics

One hundred and fourteen diabetic patients with CAD (Group I, 54 males; mean age 56.5±9.4) and 82 diabetic patients without CAD (Group II, 31 males; mean age 54.2±9.7) were included in the study. Demographic, clinical, and laboratory characteristics of the patients are presented in Table 1. There was no statistically significant difference between the groups regarding age, sex, BMI, hypertension, smoking history, white blood cell counts, hemoglobin, platelet, serum glucose, serum creatinine, HbA1c, triglyceride, total cholesterol, low-density lipoprotein cholesterol (LDL-C), high-density lipoprotein cholesterol (HDL-C) values, and medication use (p>0.05) (Table 1). EAT volumes were significantly higher in the CAD group compared to the non-CAD group (138.7±49.1 vs 98.6±34.7, p<0.001) (Figure 1).

Table 1. Baseline clinical and biochemical characteristics of study groups

Variables	Group I (N=114)	Group II (N=82)	P value
Age (years)	56.5±9.4	54.2±9.7	0.080
Male sex, n (%)	54 (47.4)	31 (37.8)	0.183
BMI (kg/m2)	29.1±4.5	27.8±4.8	0.075
Hypertension, n (%)	70 (61.4)	47 (57.3)	0.565
Smoking, n (%)	28 (24.6)	22 (26.8)	0.719
White blood cell (x103/mm3)	7.6±2.0	7.7±1.9	0.960
Hemoglobin (g/dL)	13.2±1.5	13.3±1.6	0.544
Platelet (x103/mm3)	251.5±60.7	241.9±58.3	0.269
Serum glucose (mg/dL)	147.3±50.5	148.3±62.6	0.898
Serum creatinine (mg/dL)	0.86±0.23	0.85±0.22	0.853
HbA1c (%)	7.2±1.3	7.2±1.4	0.951
Triglyceride (mg/dL)	171.4±86.1	179.9±104.1	0.533
Total cholesterol (mg/dL)	210.8±48.5	202.6±39.9	0.210
LDL- C (mg/dL)	128.4±40.3	122.8±31.5	0.292
HDL-C (mg/dL)	44.9±12.4	43.8±10.8	0.524
EAT volume (ml)	138.7±49.1	98.6±34.7	<0.001
CCTA findings			
Number of plaques	4.59±2.42		
Number of diseased segment	3.25±1.66		
Distribution of plaque subtypes			
Mixed type, n (%)	184/524 (35.1)		
Non calcified type, n (%)	157/524 (29.9)		
Calcified type, n (%)	183/524 (34.9)		
Medical treatments			
ACE inh./ARB, n (%)	55 (48.2)	29 (35.4)	0.072
B-blocker, n (%)	39 (34.2)	27 (32.9)	0.851
Statin, n (%)	29 (25.4)	19 (23.2)	0.716
Oral Antidiabetic	101 (88.6)	75 (91.4)	0.653
Insulin	42 (36.8)	27 (32.9)	0.774

Abbrevations: BMI, body mass index; LDL, low density lipoprotein cholesterol; HDL-C, high density lipoprotein cholesterol; EAT, epicardial adipose tissue; CCTA, coronary computed tomography angiography; ACE, anjiotensin converting enzyme inhibitor; ARB, angiotensin receptor blocker

In the CAD group, the number of plaques was 4.59±2.42 and the number of diseased segments was 3.25±1.66. When distribution of plaque sub-types of all 524 detected plaques was examined, the most common lesion was mixed type (n=184, 35.1%), followed by calcified type (n=183, 34.9%), and non-calcified types (n=157, 29.9%).

Association of EAT volume with clinical characteristics, laboratory findings, and coronary plaque burden and sub-types

In the correlation analysis, EAT volume was significantly correlated with male sex (r=-0.186, p=0.009), BMI (r=0.369, p<0.001), total plaque burden (r=0.424, p<0.001), mixed plaques (r=0.454, p<0.001) (Figure 2), non calcified plaques (r=0.369, p<0.001) (Figure 3) and calcified plaques (r=0.191, p=0.007) in the CAD group (Table 2). Furthermore, EAT volume was significantly positively correlated with the number of diseased segments (r=0.449, p<0.001).

Association of EAT volume with the presence of coronary artery disease

Simple logistic-regression analysis revealed that EAT volume (OR=1.023, 95%CI:1.014-1.032; p<0.001) was associated with the presence of CAD in all patients. This variable was then entered into a backward stepwise multivariate logistic-regression model. Multivariate logistic-regression analysis demonstrated that EAT volume is a significant and independent predictor for the presence of CAD in patients with DM (OR=1.023, 95%CI:1.014-1.032; p<0.001) (Table 3).

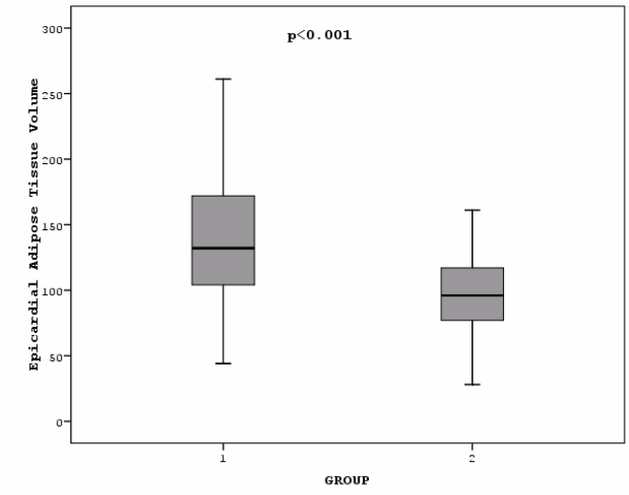

Figure 1. Epicardial adipose tissue (EAT) volume in coronary artery disease (CAD) group (Group I) and non-CAD group (Group II) in patients with diabetes mellitus

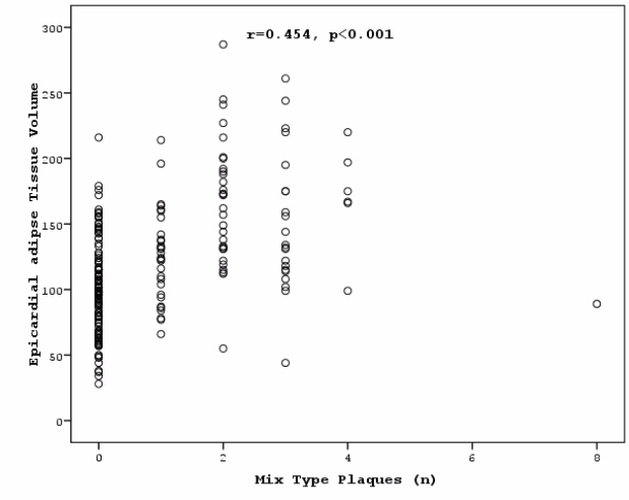

Figure 2. Correlation between epicardial adipose tissue (EAT) volume and mixed-type plaques

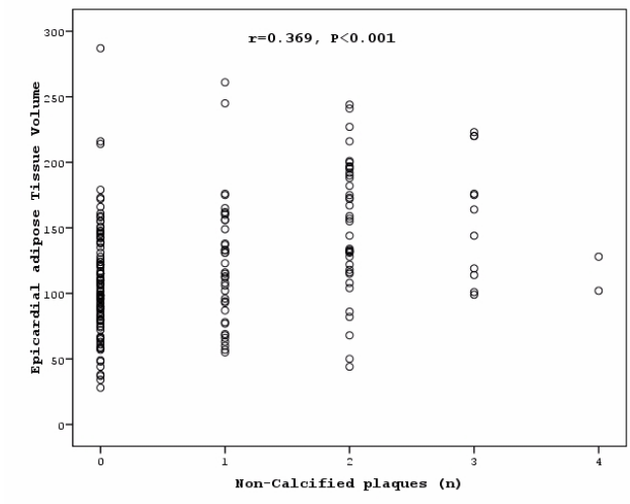

Figure 3. Correlation between epicardial adipose tissue (EAT) volume and non-calcified plaques

Table 2. Correlation analysis between Epicardial Adipose Tissue Volume and various parameters in patients with coronary artery disease

Variables	r value	p value
Age	0.127	0.077
Male sex	-0.186	0.009
BMI	0.369	<0.001
HbA1c		
	-0.053	0.464
Total plaque burden	0.424	<0.001
Mixed plaques	0.454	<0.001
Non calcified plaques	0.369	<0.001
Calcified plaques	0.191	0.007
Number of diseased segment	0.449	<0.001

Abbrevations: BMI, body mass index;

Table 3. Univariate and Multivariate regression analysis for the presence of coronary artery disease in all patients

	Univariate Logistic Regression			Multivarite Logistic Regression Analysis		
	Odds Ratio	95% CI	P Value	Odds Ratio	95% CI	P Value
Age (years)	1.030	1.000-1.062	0.053			
Male (sex)	0.710	0.399-1.264	0.245			
BMI (kg/m2)	1.059	0.995-1.126	0.070			
Serum creatinine	1.141	0.321-4.054	0.839			
LDL-C (mg/dL)	1.004	0.997-1.012	0.275			
HDL-C (mg/dL)	1.004	0.980-1.029	0.745			
HbA1c	1.015	0.818-1.261	0.891			
Hypertension	0.875	0.491-1.557	0.649			
Smoking	1.221	0.640-2.331	0.545			
EAT volume	1.023	1.014-1.032	<0.001	1.023	1.014-1.032	<0.001

Abbrevations: BMI, body mass index; LDL, low density lipoprotein cholesterol; HDL-C, high density lipoprotein cholesterol; EAT, epicardial adipose tissue.

Discussion

In the present study, we determined higher EAT volumes in diabetic patients with CAD than in non-CAD patients. Moreover, EAT volume was associated with plaque structure, and showed

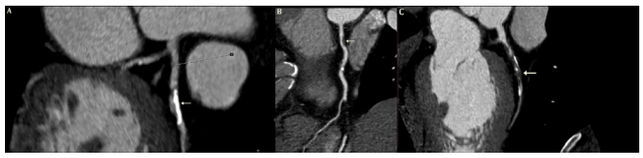

Figure 4. Sub-types of coronary plaques. Calcified plaque (A), Non-calcified plaque (B), and Mixed plaque (C) are shown with white arrow.

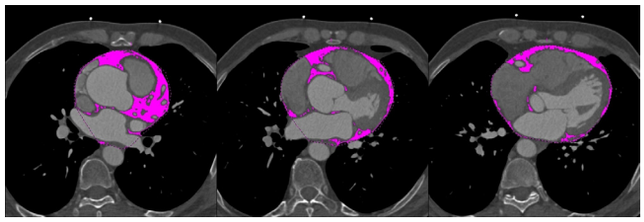

Figure 5. Quantification of the EAT volume. Segmentation of epicardial adipose tissue was achieved tracing the pericardium in the axial view (pink line). Within a smooth closed pericardial contour, the software automatically identified adipose tissue by using the threshold attenuation values of -30 to -250 HU (pink area). EAT, epicardial adipose tissue; HU, Hounsfield units.

a moderate correlation with mixed plaques and non-calcified plaques, and weak correlation with calcified plaques.

Epicardial adipose tissue is the visceral adipose tissue that is in direct contact with myocardium and coronary arteries. EAT originates from the same embryological tissue as abdominal visceral adipose tissue (VAT), namely splanchnopleuric mesoderm. By secreting various inflammatory mediators, EAT triggers the pathogenesis of atherosclerosis via its paracrine effects [2]. Studies have shown an association between EAT thickness and the presence and extent of CAD [4]. Furthermore, the myocardial ischemia-related ventricular repolarization parameters Tp-e and Tp-e/QT are also increased in patients with thicker EAT [11]. A study by Park et al. that evaluated the association between EAT thickness and plaque structure using virtual histology intravascular ultrasound (VH-IVUS) revealed that thin-cap fibroatheromas (TCFAs) and higher necrotic core burden are associated with EAT thickness [12]. It is also a fact that echocardiography, one of the most common methods for evaluating EAT, has limitations related to echocardiographic acoustic window quality and to which EAT thickness localization represents CAD risk more appropriately. In contrast, volumetric quantification of EAT using MDCT has been shown to have superior reproducibility compared to thickness and area measurements [7]. Studies that have investigated the association between EAT volume and CAD have revealed a significant correlation between the presence and extent of obstructive and non-obstructive CAD and EAT volume, as well as coronary artery calcium (CAC) score, a marker for the extent of CAD [3,13,14]. In the subanalysis of Multi-Ethnic Study of Atherosclerosis (MESA), EAT volume was associated with risk of acute myocardial infarction, resuscitated cardiac arrest, angina, and fatal coronary heart disease based upon 5 years of follow-up [5]. Similarly, in another study, increased EAT volume was directly associated with CAD and predicted major adverse cardiac events (MACE) independent of the age, gender, and conventional risk factors [15].

Recent studies have investigated the relationship between epicardial fat volume and CAD in diabetic patients. A study by Wang et al. demonstrated higher EAT volumes in type 2 diabetic patients compared to non-diabetic patients, and EAT volume was associated with components of metabolic syndrome, a CAC score which reflects the extent of CAD, and Gensini score [6]. On the other hand, a study by Groves et al. determined higher EAT volumes in type 2 diabetic patients and the higher EAT volume was associated with CAD severity. EAT volume >120 mL was emphasized as an independent predictor for the presence of significant CAD [16]. A study presented by Mohar et al. reported similar findings and showed that increased EAT volume in asymptomatic patients with Type 2 DM was associated with the presence of severe CAD, independent of BMI and CAC scores, and traditional cardiovascular risk factors [17]. Similarly, we have demonstrated higher EAT volumes in the CAD group than in the non-CAD group among diabetic patients. EAT volume was also significantly correlated with indexes of CAD severity such as total plaque burden and the number of diseased segments. Moreover, we have shown that EAT volume is an independent predictor for the presence of CAD in diabetic patients.

Coronary computed tomography angiography can provide information on both the degree of coronary stenosis and plaque morphology [8]. Another study has shown that non-calcified and mixed plaque types are more common in acute coronary syndrome (ACS) patients and the incidence of cardiac events is more common in non-calcified and mixed plaques compared to calcified plaques [18]. While the association between EAT volume and the presence of coronary atherosclerosis has been extensively studied, its association with coronary plaque morphology is still unclear. Recent studies have focused on this aspect. A study by Ding et al. has shown that EAT volume was significantly associated with the presence of calcified plaques and the Agatston CAC score [19]. Furthermore, a study by Bettencourt et al., reported that EAT volume was significantly associated with the number of coronary segments with atherosclerotic plaques and the number of segments with significant stenosis, and that increased EAT volume was an independent predictor for a higher CAC score [20]. In a prospective study that investigated the changes in coronary plaque structure and EAT volume, it was reported that increased EAT volume was a predictor for the progression of calcified plaques [21]. Likewise, in our study, EAT volume was significantly correlated with the number of diseased segments and the number of calcified plaques.

As there is a known relationship between vulnerable plaque structure and the development of ACS, the association of EAT volume with the presence of vulnerable plaques and its components has been rigorously investigated. A study by Ito et al. demonstrated that patients with CT-derived vulnerable plaques with low CT attenuation (CT density value of the plaque <30 HU) and large remodeling index (remodeling index >1.10) had higher EAT volume and that it was an independent predictor for the presence of vulnerable plaques [22]. Another study investigated the association between EAT volume and plaque characteristics. It reported that in patients with non-calcified and mixed plaques, EAT volume was higher compared to those with calcified plaques, and that it was an independent predictor for non-calcified plaques and obstructive CAD [23]. Additionaly, in a study presented by Oka et al. on the association between non-

calcified plaque features and EAT volume, it was shown that higher EAT volume is an independent predictor for vulnerable plaque components such as low-density plaque and positive remodeling [24]. The relationship between EAT volume and cardiovascular events has also been investigated. A study by Nakanishi et al. has shown that in patients followed up with MDCT, increased EAT volume was associated with increased long-term ACS outcomes [25]. Moreover, an association between EAT volume and fatal and non-fatal coronary events, independent from the cardiovascular risk factors, has been reported in recent studies [5,15]. In accordance with these studies, we have also detected a moderate correlation between EAT volume and the number of vulnerable plaques, such as mixed and non-calcified plaques. Another striking result of our study is that EAT volume showed a weak correlation with the number of calcified plaques. In our study, the positive correlation, especially with EAT volume and the vulnerable plaques, supports the association of EAT volume and long-term adverse cardiovascular outcomes.

Study Limitations

Firstly, in the present study, we could not measure serum or tissue inflammatory cytokines, adipokines, and markers such as hs-CRP all of which can lead to accelerated atherosclerosis or plaque vulnerability. Secondly, the potential incremental value of EAT measurements for cardiovascular risk prediction was also not tested, as no event follow-up was done. Finally, the study population belonged to a single ethnic group and it is unclear if our results are applicable to other ethnic groups.

Conclusion

In our study we have shown that EAT volume is an independent predictor for CAD in diabetic patients. Moreover, EAT volume showed moderate correlation with total plaque burden, number of diseased segments, and the number of mixed and non-calcified plaques. It showed a weak correlation with calcified plaques. EAT volume may be a potential alternative marker for the presence of CAD in diabetic patients and may contribute to risk assessment for coronary events and to the selection of suitable treatment strategies in diabetic patients.

Competing interests

The authors declare that they have no competing interests.

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